1: J Investig Allergol Clin Immunol 1992 Jul-Aug;2(4):205-10

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Department of Allergology and Clinical Immunology, University of Navarra, Pamplona, Spain.

We used 56 samples of heparinized blood of patients with asthma and/or allergic rhinitis, which were stimulated with Dermatophagoides pteronyssinus or gramineous pollen and anti-IgE in order to study the effect of furosemide on histamine release in basophils. At the same time, blood samples incubated in furosemide in different concentrations (2.5 microM to 1.49 mM) were also subjected to the same stimuli. Results showed a linear and dose-dependent inhibition of histamine release for antigens ($r = 0.96$; $p < 0.01$) and anti-IgE ($r = 0.95$; $p < 0.05$), reaching 95% at maximum inhibition. These results support the hypothesis that furosemide may act on mastocytes and basophils, thus inhibiting the release of histamine and other mediators.

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1: J Allergy Clin Immunol 1994 Nov;94(5):836-43 Related Articles, Books, LinkOut

J Allergy Clin Immunol

Human FcERI-IgG and humanized anti-IgE monoclonal antibody MaE11 block passive sensitization of human and rhesus monkey lung.

Saban R, Haak-Frendscho M, Zine M, Ridgway J, Gorman C, Presta LG, Bjorling D, Saban M, Jardieu P.

Department of Surgical Sciences, School of Veterinary Medicine University of Wisconsin, Madison 53706.

IgE antibodies are thought to play an important role in the induction of allergic inflammation of the bronchi. In this study we assessed the capacity of two inhibitors, FcERI-IgG, an immunoadhesin made up of the alpha chain of the high-affinity IgE receptor joined to a truncated IgG heavy chain, and MaE11, a humanized murine anti-human IgE antibody, to prevent allergen sensitization. Lung parenchyma strips from rhesus monkeys and human beings were passively sensitized for 20 hours with serum from a ragweed-sensitive patient in the presence of 0, 1-, 5-, or 10-fold concentrations of the inhibitors relative to IgE. The parenchymal strips were then suspended in a superfusion apparatus for measurement of isometric tone and collection of superfusate for histamine analysis in response to challenge with antigen E (AgE). Nonsensitized tissues did not react to AgE challenge, whereas AgE challenge of passively sensitized tissues resulted in a time-dependent parenchymal contraction and histamine release. Both FcERI-IgG and MaE11 completely abolished the AgE-induced contraction and histamine release in a dose-dependent manner. In addition, passively sensitized lung tissues failed to respond to direct challenge with either FcERI-IgG or MaE11. The results of this study suggest that FcERI-IgG and MaE11 may have important immunotherapeutic benefit for the amelioration of IgE-mediated diseases.

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